

IN THE SPECIFICATION

Please amend the specification by inserting the enclosed "Sequence Listing" after page 33 and before the Claims.

Please amend the specification by inserting the corresponding sequence identification numbers as follows:

1) On page 13, lines 15-32 please insert new paragraph:

a2
--Method: Full-length KCNQ2 cDNAs were amplified from adult human brain cDNA using the following primers (CCCCGCTGAGCCTGAG (SEQ ID NO: 10), TGTAAAAGGTCACTGCCAGG (SEQ ID NO: 11)) with the Expand Fidelity enzyme mixture (Boehringer Mannheim, Indianapolis). The KCNQ2 cDNA clone (SEQ ID NO: 3) used in the biophysical studies was identical to the KCNQ2 cDNA (SEQ ID NO: 1) isolated previously from a fetal brain cDNA library (Singh et al., 1998) except that it had a small deletion in the carboxy intracellular domain (30 amino acids from residues 417 to 446). This region is also alternatively spliced in the KCNQ2 cDNA clone described by Biervert et al. (1998). Preparation, injection of cRNA and recording from oocytes was performed at room temperature as described previously (Dixon et al. 1996). The standard extracellular recording solution contained: 82 mM NaCl, 2 mM KCl, 1.8 mM CaCl₂, 1 mM MgCl₂, and 5 mM Na-HEPES (pH 7.6). Data collection and analysis were performed using pClamp software (Axon Instruments, Foster City, CA). --

✓ 2) On page 16, lines 29-34 please insert new paragraph:

a3
--Method: Preparation of RNA, RNase protection assays and isolation of specific rat KCNQ2 (SEQ ID NO: 8) and KCNQ3 (SEQ ID NO: 6) probes were performed as described previously (Dixon and McKinnon, 1996). RNA expression was quantitated directed from dried gels with a PhosphorImager (Molecular Dynamics, Sunnyvale, CA). --

3) On page 18, lines 17-27 please insert new paragraph:

Q4

-- Full-length hKCNQ2 cDNAs were amplified from adult human brain cDNA using standard molecular biology techniques and the following primers (CCCCGCTGAGCCTGAG (SEQ ID NO: 10), TGTAAAAGGTCACTGCCAGG (SEQ ID NO: 11)) with the Expand Fidelity enzyme mixture (Boehringer Mannheim, Indianapolis, IN). The cDNA clone used in biophysical and pharmacological studies was identical to the hKCNQ2 cDNA (SEQ ID NO: 1) previously isolated from a fetal brain cDNA library by Singh, et al. (1998) except for a small deletion in the carboxy-terminal intracellular domain resulting in a 30 amino acid deletion of residues 417-446 (SEQ ID NO: 3).

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4) On page 19, lines 1-20 please insert new paragraph:

Q5

-- PCR amplification of partial rKCNQ3 cDNA clones from rat brain and rat superior cervical ganglia (SCG) cDNA was performed. An initial sequence encompassing the entire open reading frame of the rKCNQ3 gene was determined through several rounds of 5' and 3' RACE PCR using initial anchor oligonucleotides complementary to the partial cDNA clone and SCG cDNA as a template for amplification. Once cDNAs were obtained that extended beyond both the 5' and 3' ends of the open reading frame, oligonucleotides complementary to non-coding regions at either end of the coding sequence were designed. Multiple full-length cDNA clones were amplified in independent PCR reactions from rat SCG cDNA using Expand Long Template PCR (Boehringer Mannheim, Indianapolis, IN) using several combinations of the following oligonucleotides: forward (TTGACTCCCCATCCGACCT (SEQ ID NO: 12); GCCTTGCCTTCTTTGGG (SEQ ID NO: 13)), reverse (ACCGCGCACATGCATG (SEQ ID NO: 14), GTGACATGGGGAGGAAGAA (SEQ ID NO: 15)). Four independent clones were sequenced in their entirety in both directions by automatic sequencing (GenBank accession number AF091247) (SEQ ID NO: 6). --

5) On page 20, lines 19-23 please insert new paragraph:

a4 -- It is understood that one skilled in the art, using the methods taught herein and methods known in the literature, could construct a stable mammalian cell line that coexpresses KCNQ2 and KCNQ3 channels, preferably hKCNQ2 (SEQ ID NO: 2) or (SEQ ID NO: 6)) and hKCNQ3 (SEQ ID NO: 5) channels. --

6) On page 23, lines 22-36 please insert new paragraph:

a7 -- Using the method of the invention, the selective blocking of the M-channel is demonstrated in HEK 293E cells stably expressing the hKCNQ2 (SEQ ID NO: 3) potassium channel. Figure 6 illustrates that linopirdine induced a time- and concentration-dependent increase in fluorescence of HEK 293E cells stably expressing the hKCNQ2 potassium channel. These cells were loaded with the voltage-sensitive fluorescent dye, DiBAC, that distributes across cell membranes in a voltage-dependent manner. As cells depolarize (become more positive inside), more dye enters the cells and an increase in fluorescence occurs. Thus, these results indicate that under the conditions of the assay, linopirdine induced a time- and concentration-dependent depolarization which is believed to be mediated through a blockade of hKCNQ2. --

IN THE CLAIMS

Please amend Claims 5, 6, and 17, by inserting the corresponding sequence identification numbers as follows:

a8 5. The method of Claim 1 wherein KCNQ2 is hKCNQ2 (SEQ ID NO: 3).

a8 6. The method of Claim 1 wherein KCNQ3 is hKCNQ3 (SEQ ID NO: 5).

a9 17. The method of Claim 1 comprising contacting a compound with a mammalian cell that coexpresses hKCNQ2 (SEQ ID NO: 3) and hKCNQ3 (SEQ ID NO: 5), wherein the hKCNQ2